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A Nonparametric Method for Estimating Interaction Effect of Age and Period on Mortality

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In this paper we introduce a new model and develop an estimation strategy to analyze mortality data. The model we dealt with has the specific structure $E[\log q_{ij}] = \mu + \alpha_i + \beta_j + \rho_{ij}$ subject to the linear restrictions $\sum_i \alpha_i/\sigma_i^2 = \sum_j \beta_j = \sum_i \rho_{ij}/\sigma_i^2 = \sum_j \rho_{ij} = 0$ for any i and j; here, q_{ij} denotes the mortality for the jth period category and ith age category, μ denotes the overall mean, α_i denotes the ith age effect in antichronological order, β_j the jth period effect, ρ_{ij} the general interaction effect; and σ_i^2 is the common error variance of log q_{ij} for the ith age group. We propose a combined technique of ANOVA and nonparametric smoothing for estimating these parameters. The methods described are illustrated by mortality data on rectum cancer in Japanese males and females between 1950–1986.

Introduction

An age-period-cohort (APC) model or some modification of the model has often been used for analyzing mortality data. If the model is fitted correctly, then it yields useful summaries of the data in terms of parameters in the model. However, there is a well-known difficulty in estimating parameters, because of the APC model lacking identifiability (*1–4*). Setting arbitrary constraint on the parameters of the model is required to determine a unique estimate of the parameters.

To date various constraints have been proposed by many researchers (*5*). Holford (*2*) proposed that analysts concentrate their discussions only on the estimable functions such as the curvature component of each effect. Tango (*4*) analyzed Japanese mortality and detected interesting curvatures in the cohort effect, and Hirotsu (*6*) introduced a class of estimable components and discussed how to detect a systematic change in cohort effects without the class suffering from a short-term fluctuation in the context of the one-way analysis of variance. This paper introduces a new age-period model, which is free from an identifiability problem and proposes a method of model fitting to APC data through the nonparametric smoothing technique.

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Statistical Model

We derive a new model by replacing the cohort-effect term in the ordinary APC model by a term of general age × period interaction. Thus, our model is expressed as

Model I: $\log q_{ij} = \mu + \alpha_i + \beta_j + \rho_{ij} + \epsilon_{ij}$,

subject to the restrictions

$$\sum_i \alpha_i/\sigma_i^2 = \sum_j \beta_j = \sum_i \rho_{ij}/\sigma_i^2 = \sum_j \rho_{ij} = 0, \quad (1)$$

for $j = 1, \ldots, p$, $i = 1, \ldots, a$; here, q_{ij} denotes the observed mortality rate for the jth period category and the ith age category, μ is the overall mean, α_i is the fixed effect of the ith age category, β_j is the fixed effect of the jth period category, and ρ_{ij} is the fixed interaction effect associated with the ith age category and the jth period category. The only random component is ϵ_{ij}, which is assumed to be independently distributed with mean $E[\epsilon_{ij}] = 0$ and variance σ_i^2.

When there exist parameters θ and ξ such that $\rho_{ij} = \theta_i\xi_j$ for all i and j, Model I becomes the APC model attributable to James and Segal (*7*) with no cohort-effect term. Furthermore, in case of no age × period interaction, Model I is reduced to a popular two-factor age-period model (*5*). In this paper we call the reduced model age-period-main-effect mode, which is expressed as

Model II: $\log q_{ij} = \mu + \alpha_i + \beta_j + \epsilon_{ij}$.

The advantage of using Model I is that various types of age × period interaction, including the so-called co-

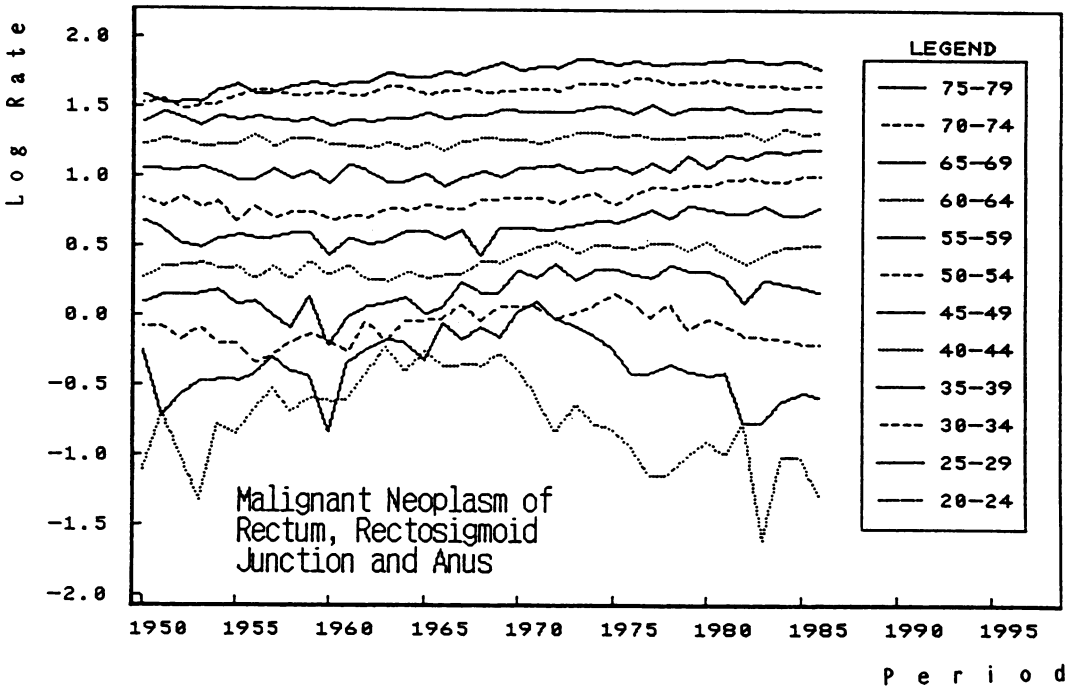

FIGURE 1. Age-specific plots of logarithmic mortality rates per 100,000 from malignant neoplasms of the rectum, rectosigmoid junction, and anus in Japanese males between 1950 and 1986.

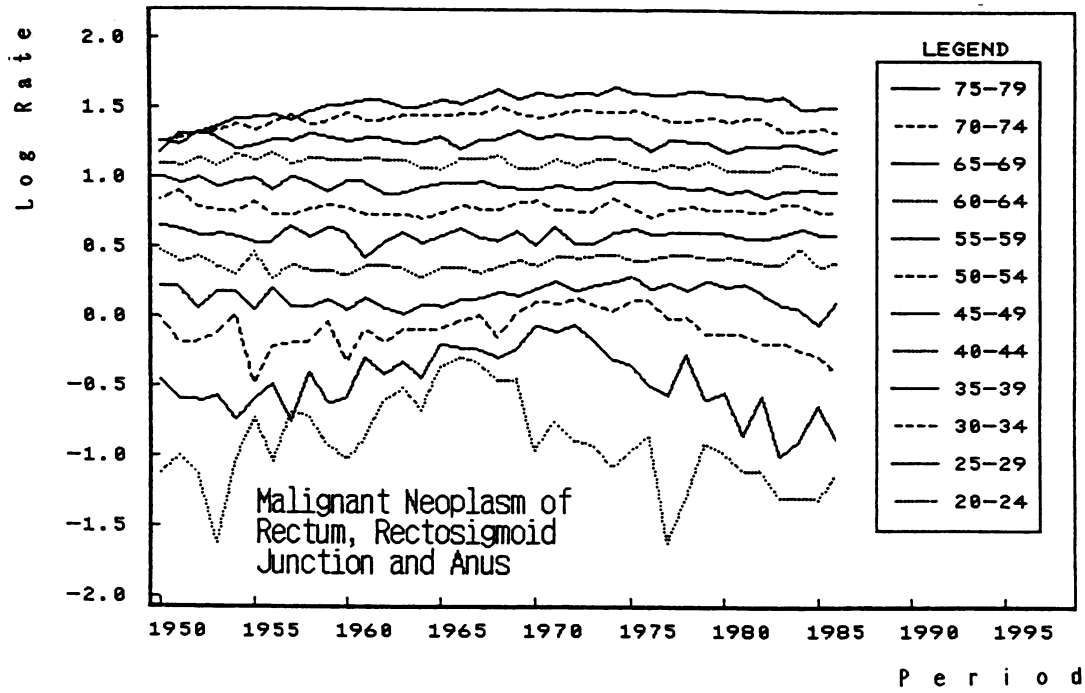

FIGURE 2. Age-specific plots of logarithmic mortality rates per 100,000 from malignant neoplasms of the rectum, rectosigmoid junction, and anus in Japanese females between 1950 and 1986.

hort effect, can be incorporated without suffering from an identifiability problem.

Estimation of Parameters

The parameters α_i and β_j of the main effects in Mode I can be estimated through ANOVA with some assumptions on the error distribution. For fitting mortality data, the Poisson error model has been the most popular and frequently used model; however, much larger deviance than degrees of freedom has been often found, even for the full model. This deviance indicates

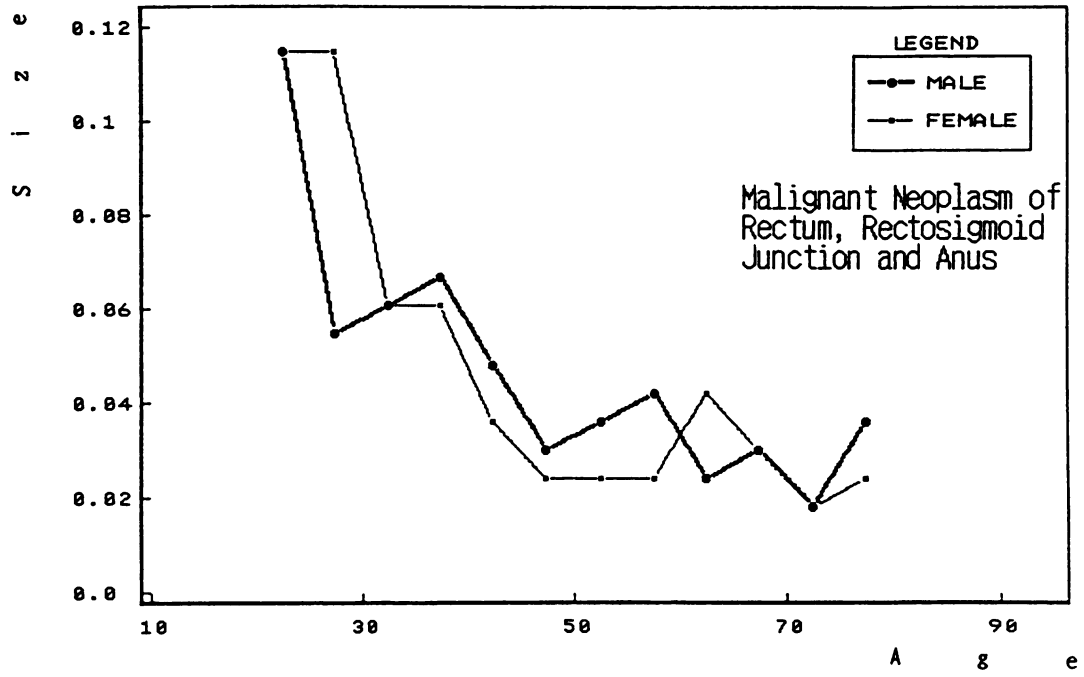

FIGURE 3. Sex-age specific plots of the square root of mean square error.

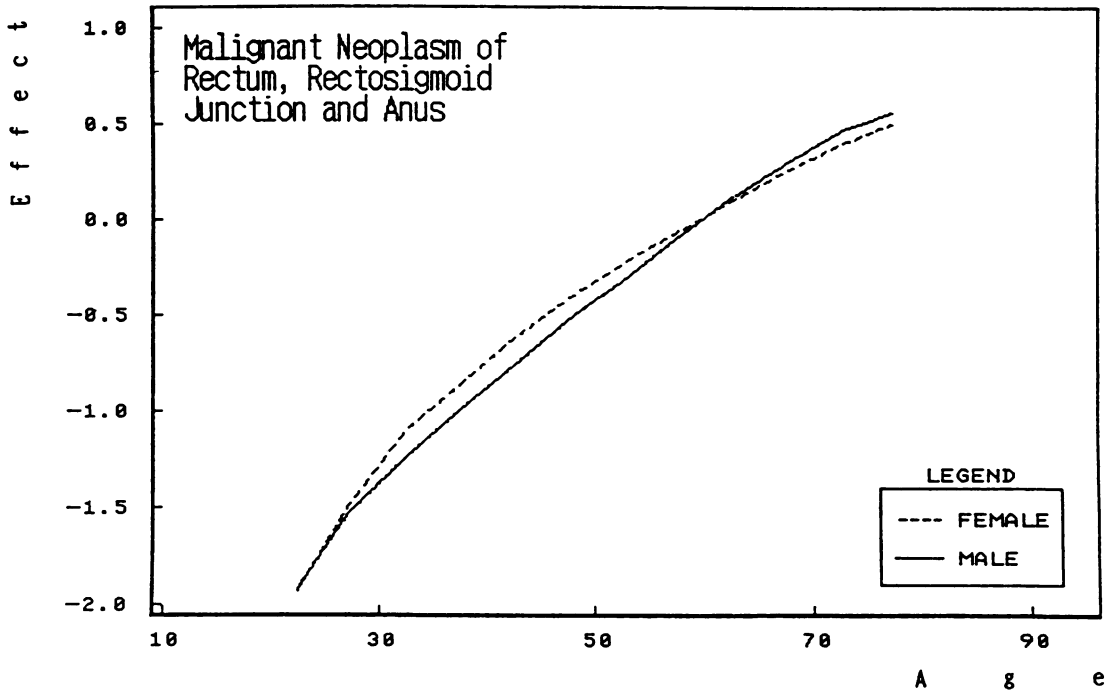

FIGURE 4. Sex-age specific plots of the estimated age-effect from Model I.

a poor fit to the Poisson distribution (*2*). In such cases, this overdispersion is referred to as extra-Poisson variation.

Breslow (*8*) proposed a general method for analyzing a data set that has extra-Poisson variation. To avoid the problem from extra-Poisson variation, we use a least-squares technique that is not based on a Poisson error model but on a more relaxed model. For many causes of death as seen in Figures 1 and 2, larger fluctuations of (logarithm of) mortality are observed in younger age groups mainly because of the Poisson variability in the number of deaths. (In fact, when the number of deaths are distributed exactly under the Poisson law, the logarithmic value of the mortality rate has the asymptotic variance, since it is the reciprocal number of the expected number of deaths. This property

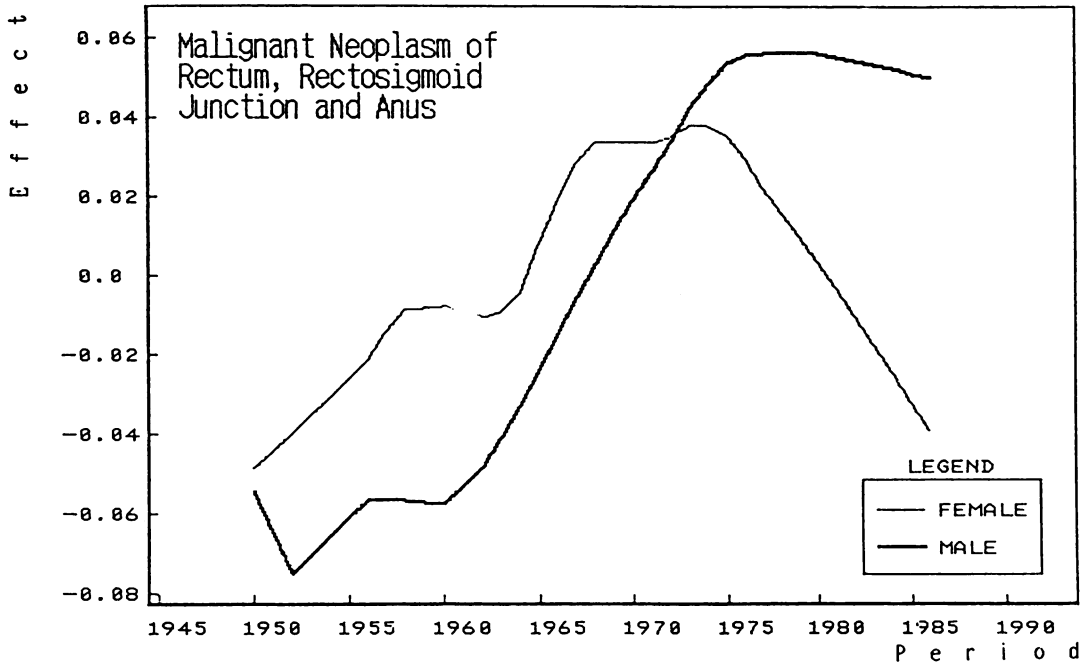

FIGURE 5. Sex-age specific plots of estimated period-effect froom Model I.

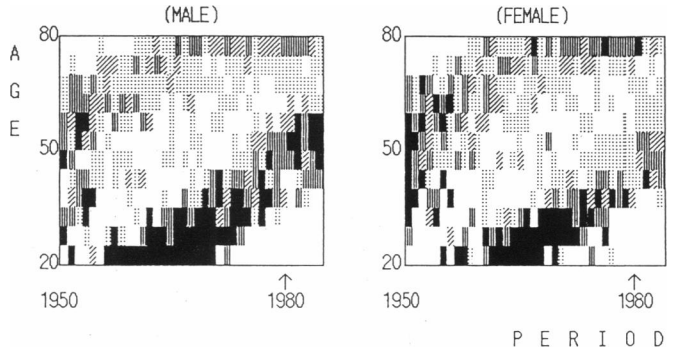

FIGURE 6. Age-period cell plots of residuals from age-period-main-effect model (Model II). In these plots, fully blackened represents $\Delta_{ij} = \text{antilog}(r_{ij}) \geq 1.2$, double shaded represents $1.1 \leq \Delta_{ij} < 1.2$, single shaded represents $1.05 \leq \Delta_{ij} < 1.1$, dotted represents $0.95 < \Delta_{ij} < 1.05$, and open represents 0.95, for each (i,j) cell.

will be referred to as Poisson variability in this paper.) Therefore, to obtain highly efficient estimates, we use a weighted least-squares technique rather than the ordinary one in estimating unknown parameters in Model I. If the weights that are proportional to the reciprocal numbers of σ_i^2 are adopted, the least-square solution is chosen to minimize the weighted residual sum of squares,

$$\sum_i (1/\sigma_i^2) \sum_j (Y_{ij} - \mu - \alpha_i - \beta_j - \rho_{ij})^2,$$

where $Y_{ij} = \log q_{ij}$, $j = 1, \ldots, p$, $i = 1, \ldots, a$. If the values of variances σ_i^2 $(i = 1, \ldots, p)$ are known, the solution $(\hat{\mu}, \hat{\alpha}^*, \hat{\beta}^*)$ could be given by

$$\begin{aligned} \hat{\mu}^* &= \tilde{Y}^*.., \\ \hat{\alpha}_i^* &= \overline{Y}_{i\cdot} - \tilde{Y}^*.., \qquad i = 1, \ldots, a, \\ \hat{\beta}_j^* &= \tilde{Y}^*._j - \tilde{Y}^*.., \qquad j = 1, \ldots, p, \end{aligned} \tag{2}$$

where $\overline{Y}_{i\cdot} = p^{-1} \sum_j Y_{ij}$, $\tilde{Y}^*._j = \sum_i c_i^* Y_{ij}$, $\tilde{Y}^*.. = p^{-1} \sum_i c_i^* \sum_j Y_{ij}$, and $c_i^* = \{\sigma_i^2 / \sum_{l=1}^{a} \sigma_l^2\}^{-1}$. Note that the solution $(\hat{\mu}^*, \hat{\alpha}^*$ and $\hat{\beta}^*)$ given by Eq. (2) will be also derived from minimizing the weighted residual sum of squares from Model II.

$$\sum_i (1/\sigma_i^2) \sum_j (Y_{ij} - \mu - \alpha_i - \beta_j)^2,$$

under the restriction of Eq. (1).

When σ_i^2 is unknown, as in the ordinary situation, substituting a suitable estimate s_i^2 for the unknown parameter σ_2^i in the weights c_i^*, we deduce the approximate least square estimates of μ, α, and β as follows

$$\begin{aligned} \hat{\mu} &= \tilde{Y}.., \\ \hat{\alpha}_i &= \overline{Y}_{i\cdot} - \tilde{Y}.., \qquad i = 1, \ldots, a, \\ \hat{\beta}_j &= \tilde{Y}._j - \tilde{Y}.., \qquad j = 1, \ldots, p, \end{aligned} \tag{3}$$

where $\tilde{Y}._j = \sum_i c_i Y_{ij}$, $\tilde{Y}.. = p^{-1} \sum_j c_i \sum_j Y_{ij}$, and $c_i = (s_i^2 / \sum_{l=1}^{a} s_l^2)^{-1}$.

As for s_i^2, one possible choice is

$$s_i^2 = 2/\{3(p-2)\} \cdot \sum_{j=2}^{p-1} \{Y_{ij} - 1/2\,(Y_{ij-1} + Y_{ij+1})\}^2. \tag{4}$$

[See Gaser et al. (*9*) or Ohtaki (*10*)].

In a situation where a smooth trend in age (or period) effect can be assumed, smoothing the sequence of $\{\hat{\alpha}_i\}$ (or $\{\hat{\beta}_j\}$) over i (or j) may yield a more plausible estimate of $\{\alpha_i\}$ (or $\{\beta_j\}$).

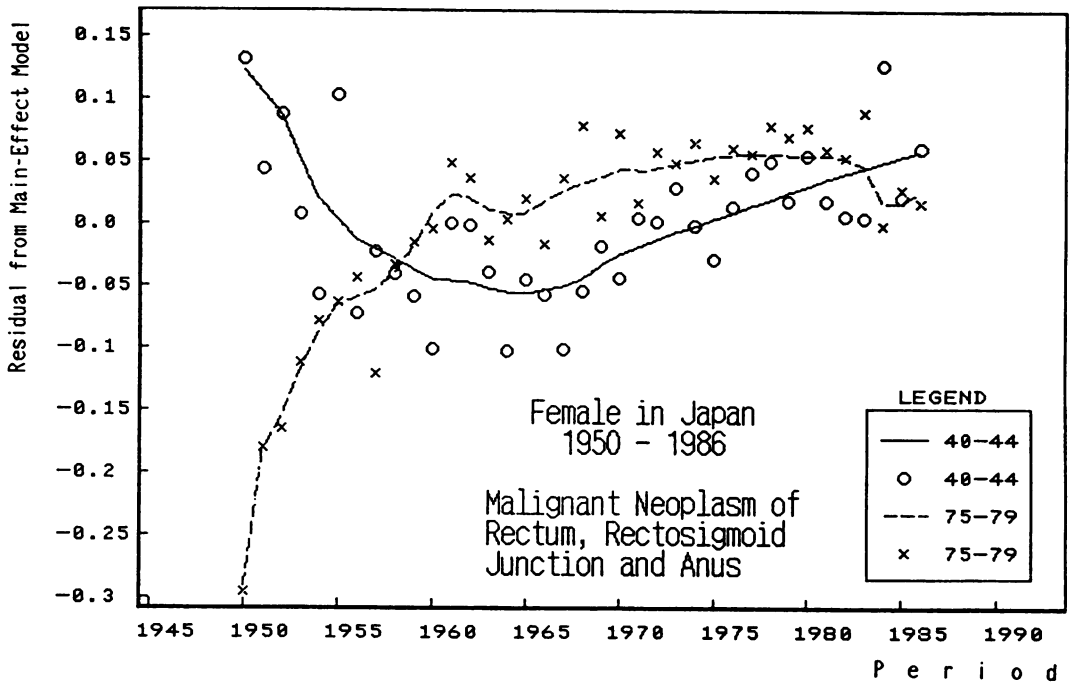

FIGURE 7. Residual plots from age-period-main-effect model (Model II) and smoothed lines.

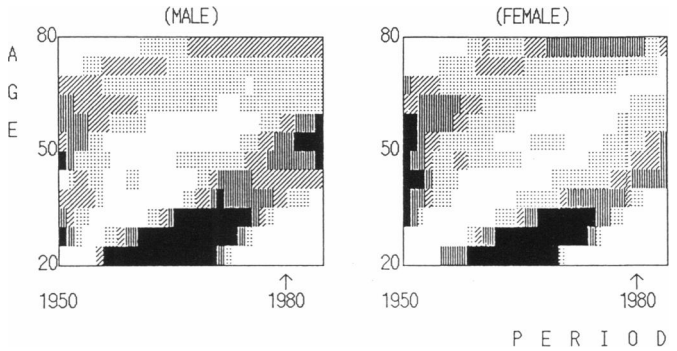

FIGURE 8. Cell plots of estimated age × period interaction effect. In these plots, fully blackened represents $\Delta_{ij} = \text{antilog}(\hat{\rho}_{ij}) \geq 1.2$, double shaded represents $1.1 \leq \Delta_{ij} < 1.2$, single shaded represents $1.05 \leq \Delta_{ij} < 1.1$, dotted represents $0.95 < \Delta_{ij} < 1.05$, and open represents $\Delta_{ij} \leq 0.95$, for each (i,j) cell.

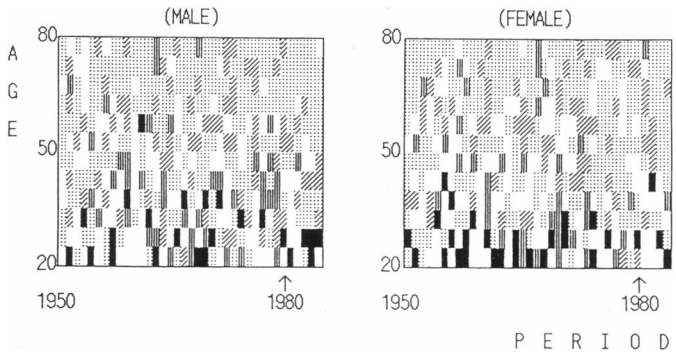

FIGURE 9. Cell plots of residuals from Model I. In these plots, fully blackened represents $\Delta_{ij} = \text{antilog}(\hat{\epsilon}_{ij}) \geq 1.2$, double shaded represents $1.1 \leq \Delta_{ij} < 1.2$, single shaded represents $1.05 \leq \Delta_{ij} < 1.1$, dotted represents $0.95 < \Delta_{ij} < 1.05$, and open represents $\Delta_{ij} \leq 0.95$, for each (i,j) cell.

On the other hand, the estimates of interaction effect, ρ_{ij}, in Model I are not available through ordinary ANOVA owing to no multiple observations in APC data. (Recently, Hirotsu (*6*) proposed an exceptional ANOVA technique to estimate the interaction effect.) Our approach to this problem is to use the nonparametric smoothing technique. Note that the residual from the age-period-main-effect model,

$$r_{ij} = Y_{ij} - \hat{\mu} - \hat{\alpha}_i - \hat{\beta}_j, \tag{5}$$

can be approximately decomposed into two components ρ_{ij} and s_{ij}—the interaction effect and the random components, respectively. Assuming that ρ_{ij} is a smooth function of j for each i, we estimate $\{\rho_{ij} | j = 1, \ldots, p\}$ through smoothing the sequence of residuals $\{r_{ij} | j = 1, \ldots, p\}$ for each $i = 1, \ldots, a$. In smoothing such sequence data, a locally linear smoother, for example, the Lowess attributable to Cleveland (*11*), will work effectively.

Illustration of Data Analysis

We now illustrate our method using real mortality data. The data concerns rectum cancer, more precisely, malignant neoplasm of the rectum, rectosigmoid junction, and anus. Mortality rates in Japanese males and females were obtained from the Japanese Vital Statistics List published from 1950 to 1986 (*12*). Figures 1

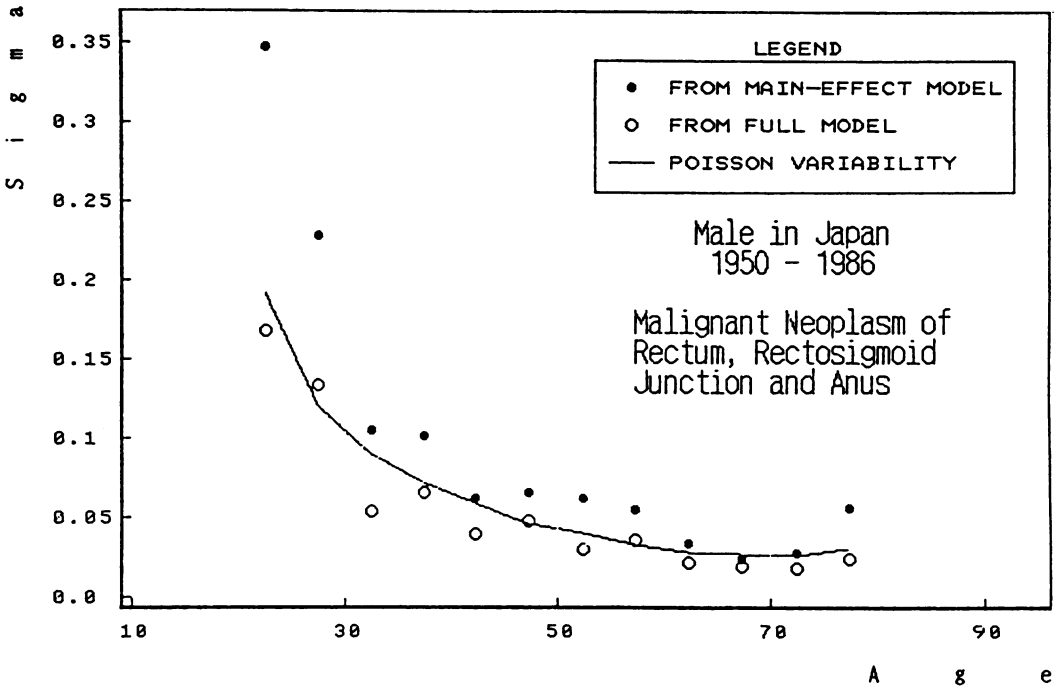

FIGURE 10. Age-specific plots of square root of mean square error and predicted values from Poisson variability on logarithmic mortality rates fitted to malignant neoplasms of rectum, rectosigmoid junction, and anus in Japanese males between 1950 and 1986.

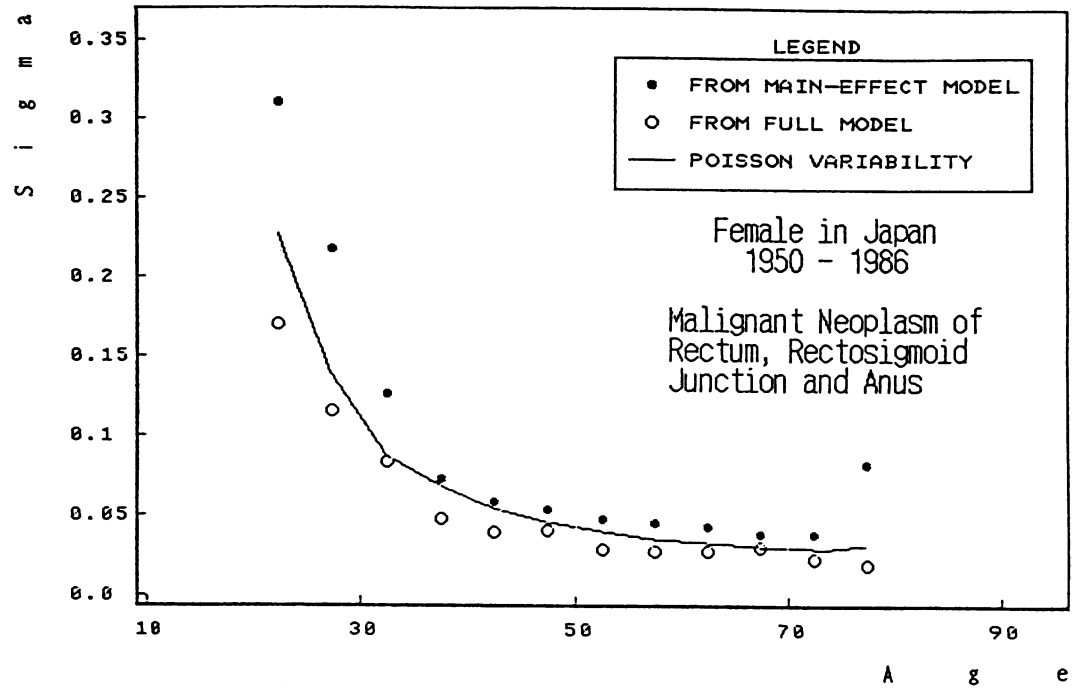

FIGURE 11. Age-specific plots of square root of mean square error and predicted values from Poisson variability on logarithmic mortality rates fitted to malignant neoplasms of rectum, rectosigmoid junction, and anus in Japanese females between 1950 and 1986.

and 2 give the age-specific plots of the logarithmic mortality rates per 100,000 for the male and female data set, respectively. These rates are based on 5-year age intervals and single-year period intervals. Figure 3 shows the sex-age-specific plots of the square root of s_i^2 calculated from Eq. (4). Briefly, a common pattern is observed in the plots for both sexes; the values of s_i^2 for the younger age groups are larger than those for middle or advanced age groups. This trend is surely the result of Poisson variability in the observed number of deaths. Figures 4 and 5 give the plots of the estimated age effects $\hat{\alpha}_i$ and period effects $\hat{\beta}_j$ computed from Eq. (3).

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According to each computed value of Δ_{ij} = antilog(r_{ij}), we produced a cell-pattern: "fully blackened" if $1.2 \leq \Delta_{ij}$, "double shaded" if $1.1 \leq \Delta_{ij} < 1.2$, "single shaded" if $1.05 \leq \Delta_{ij} < 1.1$, "dotted" if $0.95 < \Delta_{ij} < 1.05$, "whitened" if $\Delta_{ij} \leq 0.96$, for $i = 1, \ldots, a$; $j = 1, \ldots, p$.

A brief outline on the structural age × period interaction pattern can be seen in this plot. To remove the noisy fluctuation in residuals we apply a nonparametric smoothing to sequence of $\{r_{ij} | j = 1, \ldots, p\}$ for each $i = 1, \ldots, a$. Figure 7 illustrates the result of nonparametric smoothing for the female data of two groups: 40 to 44 years of age and 75 to 79 years of age. After the smoothing step, we obtained a clearer interaction pattern, as shown in Figure 8, where the same plotting rule seen in Figure 6 was adopted in making the contrast, replacing r_{ij} by the smoothed value, ρ_{ij}. In Figure 8 we can detect two different kinds of age × period interaction—one seems to be related to cohort effect and the other does not. The former is a noticeable cluster of high interaction around the births cohort who were born in 1960s. It is interesting that a strong resemblance exists between the estimated age × period interaction pattern for males and females, although the corresponding period-effect patterns are remarkably different, as shown in Figure 5. Figure 9 shows a plot of residuals $\epsilon_{ij} = r_{ij} - \hat{\rho}_{ij}$, where the same plotting rule is adopted as in Figure 6, replacing r_{ij} by ϵ_{ij}. No lack of fit of Model I is suggested from this plot. To make a further inspection of goodness of fit from this plot we computed the mean square error from Model I for each age group model. Figures 10 and 11 show the age-specific plots of square root of these (approximate) mean square errors and the predicted values from Poisson variability for the data on males and females, respectively. We can see from these plots that there is good agreement between observed value and the predicted one from Poisson variability, suggesting that no additional variation beyond Poisson variability exists in these data.

Additional Remarks

We adopt Model I as the main effect of combining age and period rather than that of age and cohort. One of the reasons why we choose the age-period-main-effect model is that the model is easier to handle than the age-cohort-main-effect model in parameter estimation. For example, since a limited amount of mortality data are available for extremely old or new cohorts, some treatment of missing values is inevitable when the age-cohort-main-effect model is adopted; such treatment is not required when the age-period-main-effect model is used. A more essential reason in our choice lies in our belief that recent improvements in medical or public health care, such as advances in developing effective medicines and introducing screening programs, are equally effective over all age groups. For such situations, we suppose that the age-period-main-effect model is more suitable than the age-cohort-main-effect model in describing mortality rates.

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